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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/980,381	03/06/2002	Huda Y. Zoghbi	HO-P01899US3(09906355)	1277

26271 7590 03/25/2005

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EXAMINER	
WILSON, MICHAEL C	

ART UNIT	PAPER NUMBER
1632	

DATE MAILED: 03/25/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/980,381	ZOGHBI ET AL.	
	Examiner	Art Unit	
	Michael C. Wilson	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *10 February 2005*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *48,55 and 57-60* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *48,55 and 57-60* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 2-1-05 has been entered.

Claims 1-47, 49-54 and 56 have been cancelled. Claims 58-60 have been added. Claims 48, 55 and 57-60 are under consideration in the instant office action as they relate to the elected subject matter: nucleic acid sequences encoding a fusion protein comprising an "atonal-associated protein". The species election of an atonal protein was withdrawn in the office action sent 4-2-04.

Applicant's arguments filed 2-1-05 have been fully considered but they are not persuasive.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

The marked up copy of the claims filed 2-1-05 has an error. The words "encoding a" in line 5 of claim 55 should have been underlined because they are new to claim 55. Please check claim amendment markings for accuracy.

Priority

Provisional application 60/176993 (1-19-00) suggested fusion proteins comprising an atonal-associated amino acid sequence operably linked to a nucleic acid sequence encoding a receptor-binding domain of a bacterial toxin (claim 103) or a nucleic acid sequence encoding a protein transduction domain (claim 104). Provisional application '993 does not support the aspect of Hath1 protein as claimed. Claims 48, 55 and 57-60 have support as they relate to Math1 fusion proteins as currently claimed to '993 (1-19-00).

Claim 50 in provisional application 60/137,060 (6-1-99) claims a "composition comprising a Math1 protein or gene in combination with a delivery vehicle". Claim 60 claims "the composition of claim 50, wherein Math1 and the receptor-binding domain of a bacterial toxin comprises a fusion protein." It is not readily apparent that the "fusion protein" in claim 60 comprises Math1 because "the receptor-binding domain of a bacterial toxin" in claim 60 lacks antecedent basis and because the bacterial toxin may as a delivery vehicle using covalent bonds and not by protein expression from a hybrid gene comprising the bacterial toxin and Math1. As written, it cannot be determined that claim 60 encompasses a hybrid gene encoding a bacterial toxin and Math1. Provisional application '060 does not support the Hath1 protein claimed or the concept of a fusion protein comprising a protein transduction domain as claimed. Claims 48, 55 and 57-60 as currently claimed do not have priority to '060.

Acknowledgment is made of applicant's claim for foreign priority based on an application filed in PCT/US00/15410 on 6-1-00. The certified copy was filed 3-6-02.

Claims 48, 55 and 57-60 have support as they relate to a nucleic acid sequence encoding a fusion protein comprising i) Math1 and ii) a bacterial toxin or a protein transduction domain as currently claimed to '993 (1-19-00).

Claims 48, 55 and 57-60 have support as currently claimed (a nucleic acid sequence encoding a fusion protein comprising i) Math1 or Hath1 and ii) a bacterial toxin or a protein transduction domain) to PCT/US00/15410 (6-1-00) (see claims 44 and 45 taken with pg 23, lines 15-18).

The effective filing date of Math1 embodiments as claimed is 1-19-00.

The effective filing date of Hath1 embodiments as claimed is 6-1-00.

Claim Rejections - 35 USC § 112

New Matter

The rejection of claims 48, 55 and 57 under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claims contain subject matter that was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventors, at the time the application was filed, had possession of the claimed invention has been withdrawn.

The new matter rejection regarding a nucleic acid sequence encoding a polypeptide that has at least about 80% identity to SEQ ID NO:58 in claims 48 and 55 has been withdrawn because the phrase has been deleted.

The new matter rejection regarding a nucleic acid sequence encoding a protein that has at least 80% identity to both SEQ ID NO:58 and 70 in claims 48 and 55 has been withdrawn because the phrase has been deleted.

Support for the transduction domain comprising HIV TAT protein in claim 57 and new claim 60 is found by piecing together the specification. Pg 10, lines 15-19, contemplates using the nucleic acid sequence in combination with a "delivery vehicle" wherein the "vehicle is the receptor-binding domain of a bacterial toxin or any fusion molecule or is a protein transduction domain. In a specific embodiment said protein transduction domain is from the HIV TAT peptide." Pg 10 does not explicitly contemplate the protein transduction domain has been fused with Math1 or is part of a fusion protein. However, pg 108, Example 22, contemplates combining an 11 amino acid "protein transduction domain" of HIV tat protein with atonal protein to make a fusion protein to allow a rapid dispersal into the nucleus of all cells of the body. Therefore, it is readily apparent that the discussion of transduction domains on pg 10 refers to a fusion protein as described on pg 108.

The phrase wherein the bacterial toxin is selected from the group consisting of exotoxin A, cholera toxin, and ricin" in new claims 58 and 59 has support on pg 72, line 19.

Written Description

Claims 55 and 57 remain rejected and claim 58 is rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claims contain subject matter that was not described in the specification in such a way as to

reasonably convey to one skilled in the relevant art that the inventors, at the time the application was filed, had possession of the claimed invention for reasons of record.

The rejection of claims 48 and 55 regarding a nucleic acid sequence encoding a polypeptide that has at least about 80% identity to SEQ ID NO:58 as broadly claimed has been withdrawn because the phrase has been deleted.

The rejection of claims 48 and 55 regarding a nucleic acid sequence encoding a protein that has at least 80% identity to both SEQ ID NO:58 and 70 has been withdrawn because the phrase has been deleted.

Support for the transduction domain comprising HIV TAT protein in claim 57 and new claim 60 is found by piecing together the specification. Pg 10, lines 15-19, contemplates using the nucleic acid sequence in combination with a "delivery vehicle" wherein the "vehicle is the receptor-binding domain of a bacterial toxin or any fusion molecule or is a protein transduction domain. In a specific embodiment said protein transduction domain is from the HIV TAT peptide." Pg 10 does not explicitly contemplate the protein transduction domain has been fused with Math1 or is part of a fusion protein. However, pg 108, Example 22, contemplates combining an 11 amino acid "protein transduction domain" of HIV tat protein with atonal protein to make a fusion protein to allow a rapid dispersal into the nucleus of all cells of the body. Therefore, it is readily apparent that the discussion of transduction domains on pg 10 refers to a fusion protein as described on pg 108.

The phrase wherein the bacterial toxin is selected from the group consisting of exotoxin A, cholera toxin, and ricin" in new claims 58 and 59 has written description on pg 72, line 19.

The rejection of claim 48 regarding a nucleic acid sequence encoding a fusion protein comprising any "amino acid sequence that is not an atonal-associated amino acid sequence" has been withdrawn because claim 48 as newly amended limits the non-atonal-associated protein to a receptor binding domain of a bacterial toxin or a protein transduction domain. The specification describes a fusion protein comprising Math1 or Hath1 and a bacterial toxin or a transduction domain on pg 10, lines 15-19, in view of pg 108, Example 22, and in the description of a fusion protein comprising a bacterial toxin or a transduction domain as a delivery vehicle on pg 10, lines 15-19. It would be readily apparent from pg 10, lines 15-19, that transduction domain on pg 108, Example 22, could be replaced with a bacterial toxin. It is noted that pg 72, lines 17-19, states bacterial toxins can be used as delivery vehicles, such as Exotoxin A, cholera toxin and Ricin toxin.

The rejection of claim 55 regarding a nucleic acid sequence encoding a fusion protein comprising any "nucleic acid sequence that is not an atonal-associated nucleic acid sequence" has been withdrawn because the phrase has been deleted in claim 55 as newly amended.

Claim 55 remains rejected under written description. The claim requires a composition comprising a nucleic acid sequence encoding Math1 or Hath1 and a delivery vehicle, further comprising a nucleic acid sequence encoding a receptor binding

domain of a bacterial toxin or a protein transduction domain. As written, claim 55 encompasses a composition in which the nucleic acid sequence encoding the bacterial toxin or transduction domain is not part of the nucleic acid sequence encoding Math1 or Hath1. However, pg 10, lines 15-19, in view of pg 108, Example 22, is limited to delivering a nucleic acid sequence encoding a fusion protein, i.e. a protein formed by the expression of a hybrid gene (see definition of "fusion protein" by On-line Medical Dictionary). Therefore, the specification is limited to a composition comprising a fusion gene encoding i) Math1 or Hath1 and ii) a receptor binding domain of a bacterial toxin or a protein transduction domain two proteins. The specification does not contemplate a composition comprising two separate nucleic acid sequences as broadly encompassed by claim 55 as newly amended. Claiming such breadth of compositions without contemplating such a breadth, describing the structure or function of compositions is not in compliance with the description requirement. Rather, it is an attempt to preempt the future before it has arrived (See *Fiers v. Revel*, 25 USPQ2d 1601 (CA FC 1993) and *Regents of the Univ. Calif. v. Eli Lilly & Co.*, 43 USPQ2d 1398 (CA FC, 1997)). Claims 57 and 58 are included because they are dependent upon claim 55.

Enablement

Claims 55 and 57 remain rejected and claim 58 is rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a nucleic acid sequence encoding a fusion protein, said fusion protein comprising a Math1 protein that is at least about 80% identical to SEQ ID NO:58 operably linked to a bacterial toxin or to a protein transduction domain, does not reasonably provide enablement for a nucleic

acid sequence encoding any atonal-associated protein that is at least about 80% identical to SEQ ID NO:58 as broadly claimed, or that is at least about 80% identical to both SEQ ID NO:58 and SEQ ID NO:70 operably linked to a protein transduction domain of HIV tat protein. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention commensurate in scope with these claims.

The rejection regarding using a nucleic acid sequence encoding a polypeptide that has at least about 80% identity to SEQ ID NO:58 as broadly claimed in claims 48 and 55 has been withdrawn because the limitation has been deleted.

The rejection of claim 48 regarding how to use a nucleic acid sequence encoding a fusion protein comprising Math1 and any "amino acid sequence that is not an atonal-associated amino acid sequence" has been deleted because the non-atonal-associated amino acid sequence has been limited to a receptor binding domain of a bacterial toxin or a protein transduction domain.

The rejection of claim 55 regarding how to use a nucleic acid sequence encoding a fusion protein comprising any "nucleic acid sequence that is not an atonal-associated nucleic acid sequence" has been withdrawn because the phrase has been deleted as newly amended.

The rejection of claim 55 regarding how to use a composition comprising a nucleic acid sequence encoding i) Math1 or Hath1, further comprising a nucleic acid sequence encoding a receptor binding domain of a bacterial toxin or a protein transduction domain as broadly claimed is maintained. The specification taught a

composition comprising a fusion gene encoding Math1 or Hath1 and a transduction domain or bacterial toxin would deliver the Math1 or Hath1 protein to cells by binding the toxin or transduction domain binding via the cell membranes. If the Math1/Hath1 is not bound to the toxin or transduction domain as encompassed by claim 55, the specification not teach how to deliver the Math1 or Hath1 protein to the cell or the purpose of including the nucleic acid sequence encoding the toxin or transduction domain. One of skill could have made the composition as broadly claimed but would not have been able to use the nucleic acid sequence encoding the toxin or transduction domain to deliver Math1 or Hath1 proteins to cells. Without such guidance it would have required one of skill undue experimentation to use two separate nucleic acid sequences as encompassed by the claim. Therefore, claim 55 should be limited to a composition comprising a nucleic acid sequence encoding a fusion protein comprising i) Math1 or Hath1 and ii) a receptor binding domain of a bacterial toxin or a protein transduction domain.

Applicants argue the specification teaches how to deliver genes; therefore, applicants conclude one of skill could use the composition of claim 55 as written. Applicants' argument is not persuasive because the nucleic acid sequence encoding the receptor binding domain of a bacterial toxin or a protein transduction domain would not deliver Math1 or Hath1 if the nucleic acid sequence was delivered separately. The only use for a nucleic acid sequence encoding a bacterial toxin or a transduction domain described in the specification is as a delivery vehicle. The specification fails to teach

how to deliver Math1 or Hath1 using a nucleic acid sequence encoding a bacterial toxin or a transduction domain that is not part of a fusion gene.

The rejection of claims 48, 55 and 57 under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention has been withdrawn.

The rejection of claims 48 and 55 as they relate to non-atonal-associated amino acid sequences has been withdrawn in view of the claims as amended. Claim 48 requires "an amino acid sequence that is not an atonal-associated amino acid sequence, wherein the amino acid sequence that is not an atonal-associate amino acid sequence comprises a receptor binding domain of a bacterial toxin or a protein transduction domain." Claim 55 requires "an additional nucleic acid sequence encoding a receptor binding domain of a bacterial toxin or a protein transduction domain."

Claim Objections

Claim 48 as newly amended are objected to because it does not clearly set forth the second portion of the fusion protein. The phrase "an amino acid sequence that is not an atonal-associated amino acid sequence, wherein the amino acid sequence that is not an atonal-associated amino acid sequence comprises a receptor binding domain of a bacterial toxin or a protein transduction domain" is wordy and should be written more succinctly. For example "an amino acid sequence encoding a receptor binding domain of a bacterial toxin or a protein transduction domain" would be more succinct.

In its entirety, claim 48 may be simplified to a "nucleic acid sequence encoding a fusion protein, said fusion protein comprising i) Math1 or Hath1, and ii) a receptor binding domain of a bacterial toxin or a protein transduction domain.

Claim Rejections - 35 USC § 103

Claims 48, 55 and 57 remain rejected and claim 60 is rejected under 35 U.S.C. 103(a) as being unpatentable over Akazawa (J. Biol. Chem., 1995, Vol. 270, No. 15, pg 8730-8738) in view of Schwartze (Science, Sept. 1999, Vol. 285, pg 1569-1572) for reasons of record.

Akazawa taught transfecting eukaryotic cells with a vector encoding mouse atonal protein 1 (math1) (pg 8734, col. 2). Math1 is 87.4% identical to SEQ ID NO:58. Amino acids 160-180 of the math1 taught by Akazawa are 100% identical to SEQ ID NO:70. Akazawa did not teach the vector encoded a fusion protein comprising math1.

However, Schwarze taught a nucleic acid sequence encoding β-gal operably linked to an HIV tat protein transduction domain (¶ bridging pg 1569-1570).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to deliver DNA encoding math1 to a cell using a vector as taught by Akazawa using DNA encoding TAT as taught by Schwartze fused to the DNA encoding math1. One of ordinary skill in the art at the time the invention was made would have been motivated to add the DNA encoding TAT to the DNA encoding math1 to dramatically enhance transduction potential in cultured cells (Schwarze, pg 1572, lines 1-4). Fifty proteins ranging in size from 15-120 kD were transduced in a wide variety of

human and murine cell types using the HIV tat protein transduction domain (pg 1570, col. 2, lines 12-16).

Applicants argue one of ordinary skill would not have been motivated to use the TAT transduction domain described by Schwarze because Akazawa obtained sufficient transcription activation. Applicants' argument is not persuasive because Schwarze taught TAT improved transduction potential.

Applicants argue one of ordinary skill would not have been motivated to improve delivery of an already functional composition. Applicants' argument is unfounded. It was readily apparent to those of ordinary skill in the art that improved delivery of proteins into cells was desired as evidenced by Schwartze.

Applicants argue Akazawa did not teach making a fusion protein. Applicants' argument is moot. Akazawa has not been relied upon as teaching a Math1 fusion protein and need not teach all the limitations of the claims. Schwarze fused the TAT transduction domain to 50 proteins and has been relied upon for the limitation of fusion protein.

Applicants argue that combining TAT described by Schwarze with Math1 as described by Akazawa was inappropriate for the purposes of Akazawa. Applicants' argument is not persuasive because it is not based in any logic or evidence.

Claims 48, 55 and 57 remain rejected and claim 60 is rejected under 35 U.S.C. 103(a) as being unpatentable over Ben-Arie (Hum. Mol. Genet. 1996, Vol. 5, pg 1207-

1216) in view of Schwartze (Science, Sept. 1999, Vol. 285, pg 1569-1572) for reasons of record.

Ben-Arie taught transfecting eukaryotic cells with a vector encoding Hath1 (pg 1208, col. 1, lines 9-12, the sentence bridging col. 1-2, and col. 2, lines 1-2; pg 1213, ¶ bridging col. 1-2; pg 1215, col. 1, Physical Mapping of HATH1). Ben-Arie did not teach the vector encoded a fusion protein comprising Hath1.

However, Schwarze taught a nucleic acid sequence encoding β-gal operably linked to an HIV tat protein transduction domain (¶ bridging pg 1569-1570).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to deliver DNA encoding Hath1 to a cell using a vector as taught by Ben-Arie using DNA encoding TAT as taught by Schwartze fused to the DNA encoding math1. One of ordinary skill in the art at the time the invention was made would have been motivated to add the DNA encoding TAT to the DNA encoding Hath1 to dramatically enhance transduction potential in cultured cells (Schwarze, pg 1572, lines 1-4). Fifty proteins ranging in size from 15-120 kD were transduced in a wide variety of human and murine cell types using the HIV tat protein transduction domain (pg 1570, col. 2, lines 12-16).

Applicants argue Ben-Arie did not teach transfecting eukaryotic cells with a vector encoding Hath1. applicants argue the experiments of Ben-Arie were extracellular. applicants' arguments are not persuasive because it is unfounded and does not address the specific teachings of Ben-Arie. Sequencing requires transfection of prokaryotic cells to produce adequate amounts of Hath1 DNA for sequence analysis.

Chromosomal mapping requires transfection of eukaryotic cells with the DNA for detection of the chromosome.

Applicants argue Ben-Arie did not teach making a fusion protein. Applicants' argument is moot. Ben-Arie has not been relied upon as teaching a fusion protein and need not teach all the limitations of the claims. Schwarze has been relied upon for the limitation of fusion protein.

Applicants argue one of ordinary skill would not have been motivated to make a fusion protein of the Hath1 protein taught by Ben-Arie. Applicants' argument is unfounded. One of ordinary skill would have been motivated to make the Hath1 protein a TAT fusion protein to improve delivery. It was readily apparent to those of ordinary skill in the art that improved delivery of proteins into cells was desired as evidenced by Schwartze.

Double Patenting

The provisional double patenting rejection of claims 48, 55 and 57 conflicting with claims 112 and 117 is no longer provisional because the application has been allowed.

Applicants are reminded that a request to address a provisional rejection upon allowance of another application is non-responsive because every rejection (even provisional rejections) must be addressed. The proper response to a provisional double patenting rejection is either an argument or a terminal disclaimer. A request to hold a provisional rejection in abeyance is "non-responsive."

Claims 48, 55 and 57 and new claim 60 are rejected under the judicially created doctrine of double patenting over claim 12 (dependent upon claim 9) of U. S. Patent No. 6,838,444 since the claims, if allowed, would improperly extend the "right to exclude" already granted in the patent.

The subject matter claimed in the instant application is fully disclosed in the patent and is covered by the patent since the patent and the application are claiming common subject matter, as follows:

Claims 48 and 55 overlap with claim 12 because claim 12 requires a nucleic acid sequence encoding SEQ ID NO:58 and 70 (Math1 and Hath1 proteins) operably linked to a nucleic acid sequence encoding a transduction domain. The limitations HIV Tat in claims 57 and 60 is a species of transduction domain in claim 12 of '444 and could have been claimed in the patented application as it was a species described in the application of '444. A nucleic acid sequence encoding Math1 or Hath1 operably linked to a bacterial toxin was part of the disclosure for '444 (col. 34, lines 46-67) and was an obvious variant of the nucleic acid sequence encoding Math1 or Hath2 operably linked to a transduction domain in claim 12 of '444.

There is no apparent reason why applicant was prevented from presenting claims corresponding to those of the instant application during prosecution of the application which matured into a patent. See *In re Schneller*, 397 F.2d 350, 158 USPQ 210 (CCPA 1968). See also MPEP § 804.

Conclusion

No claim is allowed.

Inquiry concerning this communication or earlier communications from the examiner should be directed to Michael C. Wilson who can normally be reached at the office on Monday, Tuesday, Thursday and Friday from 9:30 am to 6:00 pm at 571-272-0738.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

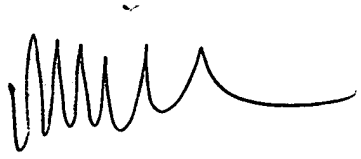
Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance. Representatives are available to answer your questions daily from 6 am to midnight (EST). The toll free number is (866) 217-9197. When calling please have your application serial or patent number, the type of document you are having an image problem with, the number of pages and the specific nature of the problem. The Patent Electronic Business Center will notify applicants of the resolution of the problem within 5-7 business days. Applicants can also check PAIR to confirm that the problem has been corrected. The USPTO's Patent Electronic Business Center is a complete service center supporting all patent business on the Internet. The USPTO's PAIR system provides Internet-based access to patent application status and history information. It also enables applicants to view the scanned images of their own application file folder(s) as well as general patent information available to the public.

For all other customer support, please call the USPTO Call Center (UCC) at 800-786-9199.

If attempts to reach the examiner are unsuccessful, the examiner's supervisor, Ram Shukla, can be reached on 571-272-0735.

The official fax number for this Group is (571) 273-8300.

Michael C. Wilson



MICHAEL WILSON
PRIMARY EXAMINER